CASE REPORT JSLS

Portal Vein Thrombosis After Laparoscopic Cholecystectomy

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ABSTRACT

Introduction: Portal vein thrombosis (PVT) is a relatively uncommon complication after abdominal surgery.

Case Report: We report an even more unusual case of PVT 10 days after an uncomplicated laparoscopic cholecystectomy, believed to be only the fourth reported case in the literature of this rare complication.

Conclusion: Albeit extremely rare, PVT should be included in the differential diagnosis for abdominal symptoms and/or elevated hepatic function tests after laparoscopic cholecystectomy.

Key Words: Portal vein, Thrombosis, Cholecystectomy, Laparoscopic surgery.

INTRODUCTION

Portal vein thrombosis (PVT) is an extremely rare complication after abdominal surgery. We report what is believed to be the fourth reported case and youngest reported patient with left PVT after laparoscopic cholecystectomy (LC). PVT after laparoscopic surgery is extremely rare but should be included in the differential diagnosis of unexplained abdominal pain after laparoscopic cholecystectomy.

CASE REPORT

The patient was a 31-year-old woman who presented initially to our service with a 6-month history of recurrent postprandial right upper quadrant pain. Her past medical history was remarkable for hypertension (which developed during pregnancy) and thyroid nodules, and she had undergone cesarean section 7 months previously. She had no remarkable family history of hypercoagulable state or thrombotic events. She was taking metoprolol and methyldopa daily to treat her hypertension. She was not taking oral contraceptive pills. Abdominal ultrasonography showed cholelithiasis without other abnormal findings. Her preoperative liver function tests showed normal transaminases (alanine aminotransferase 20 U/L, aspartate aminotransferase 13 U/L), alkaline phosphatase (95 U/L), and total bilirubin (0.3 mg/dL). Preoperative coagulation parameters were all normal. She underwent an uneventful elective laparoscopic cholecystectomy and was discharged home on the same day. Pathologic results were consistent with chronic cholecystitis.

At postoperative day 10, the patient re-presented to our emergency center complaining of right flank pain and nausea. She was afebrile and nontachycardic. Physical examination demonstrated minimal abdominal tenderness. Her laboratory results were remarkable for elevated transaminases (alanine aminotransferase 271 U/L, aspartate aminotransferase 466 U/L), alkaline phosphatase (219 U/L), and total bilirubin (1.1 mg/dL). Abdominal ultrasonography revealed, somewhat unexpectedly, left PVT **(Figure 1)**, which was confirmed with a computed tomography (CT) scan of the abdomen **(Figure 2)**. She was

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Figure 1. Ultrasound demonstrating left PVT (arrow).

Figure 2. Computed tomography of the abdomen demonstrating left PVT (arrow).

admitted to our service. A hypercoagulable workup demonstrated a low protein S (35%, range 60–120). Her protein C was normal. Anticardiolipin antibiodies (IgA, IgG, and IgM) were all within normal limits. There was no evidence of a factor II (prothrombin) G20210A mutation, factor V Leiden mutation, or JAK2 V617F mutation. She was started on therapeutic low-molecular-weight heparin (LMWH, enoxaparin). She responded to the treatment with improved liver function tests and resolved abdominal symptoms. She was discharged in good condition on hospital day 3 with therapeutic LMWH and oral warfarin and was expected to continue taking therapeutic anticoagulation medication.

Table 1.
Review of All Reported Cases* of PVT After Laparoscopic Cholecystectomy

Report	Age/Gender	Indication	Risk Factors	Coagulopathy	Presentation	Findings	Treatment
Ourania et al., 2005	32/F	Gallbladder polyps	Oral contraceptive pills	Elevated anticardiolipin antibody (IgG)	POD 7: abdominal pain and ileus	Superior mesenteric vein	Transhepatic portal vein thrombectomy, heparin Preoperative: SCD, unfractionated heparin
Dilip et al., 2011	63/F	Chronic cholecystitis	Diabetes, coronary artery disease	Dengue viral infection	POD 5: nausea, emesis, and diarrhea	Main portal vein	Intravenous hydration, antibiotics Preoperative: unknown
Waheed et al., 2012	55/F	Chronic cholecystitis	None	Factor II (prothrombin) G20210A mutation	2 months postoperatively: abdominal pain	Portal/splenic vein	Low-molecular-weight heparin Preoperative: unknown
Current Report	31/F	Chronic cholecystitis	Hypertension	Low protein S	POD 10: abdominal pain and nausea	Left portal vein	Low-molecular-weight heparin and warfarin Preoperative: SCD

POD = postoperative day; SCD = sequential compression devices.
*Includes the present report.

DISCUSSION

PVT is a recognized complication after laparoscopic procedures including Roux-en-Y gastric bypass, Nissen fundoplication, partial colectomy, cholecystectomy, and appendectomy.[1] Although rare, there are three cases reported in the English language literature of PVT after laparoscopic cholecystectomy.[2–4] All patients, including our patient, underwent what was described as uncomplicated laparoscopic cholecystectomy.

Formation of PVT in these patients is likely multifactorial. Pneumoperitoneum and the resultant increased intra-abdominal pressure has been shown to result in changes in coagulation parameters,[2] as manifested by changes in prothrombin time, international normalized ratio, D-dimer, fibrinogen, and fibrin degradation products.[5] Pneumoperitoneum also affects splanchnic and portal venous blood flow.[2] One study demonstrated that the diameter of the portal venous trunk and the mean portal blood flow were significantly decreased with pneumoperitoneum of >10 mm Hg.[6] Patient positioning during laparoscopic surgery may also affect portal blood flow and stasis.[1] Direct injury to the portal venous system may also be a contributing factor but is unlikely in our case because of the anatomy of the portal vein as a posterior structure. Another significant factor in PVT is underlying coagulopathy and underlying patient disease processes. All previously reported cases have demonstrated some hypercoagulable state **(Table 1).** Our patient had low protein S, which may have contributed to her hyperocoagulable state. It is important to note, however, that in some cases, low protein S may be associated with acute thrombosis and not be reflective of a true hypercoagulable state.[7] In all cases, the contributing coagulopathy was not detected preoperatively.

The diagnosis of PVT can be established with ultrasonography or computed tomography with intravenous contrast, both commonly used in the workup of unexplained pain and symptoms after laparoscopic cholecystectomy. Other adjuncts, including angiography and magnetic resonance imaging, may also be used. The treatment varies depending on the extent of the thrombosis and the presence or absence of bowel ischemia. It is unknown whether preoperative antithrombotic prophylaxis affects the incidence of PVT.[1] One patient required transhepatic portal vein thrombectomy for significant thrombosis, but most patients who present with PVT after laparoscopic procedures require only supportive treatment and anticoagulation. Our patient promptly improved after initiation of LMWH and was discharged on hospital day 3 with an LMWH-to-warfarin bridge. The optimal duration of anticoagulation for these patients is unknown.[3] If the patient has primary coagulation disorders, lifelong anticoagulation is required. For those with no identifiable risk factors, some suggest it is reasonable to re-image the patient after approximately 6 months of anticoagulation and stop if resolution of the thrombosis is documented.

CONCLUSION

Albeit extremely rare, PVT should be included in the differential diagnosis for abdominal symptoms and/or elevated hepatic function tests after laparoscopic cholecystectomy.

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